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STUDY OF THE RELATIONSHIP BETWEEN CARBOXYHEMOGLOBIN ON ADMISSION TO THE SUBSEQUENT HOSPITAL COURSE OF PATIENTS ADMITTED TO THE MYOCARDIAL INFARCTION RESEARCH UNIT AT THE JOHNS HOPKINS HOSPITAL

Coordinating Research Council, Incorporated

August 1975

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STUDY OF THE RELATIONSHIP BETWEEN CARBOXYHEMOGLOBIN ON ADMISSION TO THE SUBSEQUENT HOSPITAL COURSE OF PATIENTS ADMITTED TO THE MYOCARDIAL INFARCTION RESEARCH UNIT AT THE JOHNS HOPKINS HOSPITAL

On the basis of current knowledge, chronic exposure to carbon monoxide could influence the onset or severity of a myocardial infarction by two main mechanisms. First, animal experiments have indicated that the atheromatous process can be accelerated by sustained high carboxyhemoglobin (COHb) levels. Second, once a coronary occlusion occurs, the severity of ischemia could be increased by the presence of carbon monoxide even in relatively low concentrations. There is very little evidence in man supporting these hypotheses, however. This research was undertaken to investigate patients admitted to the Johns Hopkins Hospital Emergency Ward with symptoms of chest pain, to determine if carbon monoxide at admission might relate to the severity of the infarct. The possibility that chronic exposure might play a role in the severity was also evaluated.

Previously Johns Hopkins issued a final report on the studies relating carboxyhemoglobin and sudden death in Baltimore.

Patients Studied

During a 27 month period, 152 patients referred to the medical service complaining of chest discomfort were seen in the Johns Hopkins Hospital Emergency Ward, and blood samples were obtained on some of these on admissions. These 152 patients will be referred to subsequently as the EW patients. In addition, blood samples were obtained from 113 patients undergoing hemodynamic assessment within the coronary care unit during a 29 month period. These 113 patients will be referred to subsequently as the MIRU (Myocardial Infarction Research Unit) patients. Some of the patients are present in both population groups. Patients were divided into three groups (1) those with evidence of acute myocardial infarction (2) those without evidence of acute myocardial infarction and (3) those in whom the diagnosis was uncertain.

The criteria for the diagnosis of acute myocardial infarction utilized in this study were a rise in the total CPK to greater than 50 IU (upper limits of normal 32 IU) in samples obtained every six hours for the initial 48 hours of hospitalization followed by a subsequent distinct fall in the CPK level within 72 hours of admission and either (1) clearly defined historical information suggestive of acute myocardial ischemia during the 24 hour period prior to hospitalization or (2) appearance of new Q waves, a decrease in R wave potential, or evolutionary ST-T wave changes on serial electrocardiograms. The patients in the non-infarct group did not meet any of these criteria or had changes in the CPK clearly explainable on the basis of

other diagnoses aside from acute myocardial infarction. The patients in the uncertain group were those who met some but not all of the criteria for myocardial infarction outlined above. There were 140 patients in the infarct group, 52 patients in the no infarct group and 14 patients in the uncertain group.

Carbon monoxide and thiocyanate levels were determined on all samples using the methods of Small et al (1) and Bowler (2). Thiocyanate is a metabolic product of cyanide inhaled during cigarette smoking. Thiocyanate may also appear in the blood by virtue of ingestion of certain leafy vegetables. Previous data in our laboratory has suggested the following concerning serum thiocyanate levels: (1) the half-time for elimination of thiocyanate in smokers is approximately 8 days; (2) the level of thiocyanate in non-smokers seldom exceeds 4.0μg/mℓ. This last conclusion is supported in the present study since only one of the non-smokers had a thiocyanate level of greater than 4.0 μg/mℓ. Thiocyanate is a better index of smoking exposure than COHb in ill patients because unlike CO, the blood concentration is unaffected if smoking patterns change a few hours before admission. Our previous work has shown that smoking is the principal source of CO in the Baltimore population.

Smoking histories were obtained by either physician-investigators or by specially trained personnel involved in this study. From these data patients with acute myocardial infarction were divided into three population groups. (1) Non-smokers: Patients who stated that they had never smoked and had thiocyanate levels of <4.0 μg/mℓ thiocyanate level. (2) Ex-smokers: Patients with a history of greater than 20 pack years of cigarette smoking and thiocyanate level of< 4.0 μg/mℓ and (3) Recent Smokers: Patients with thiocyanate levels of >4.0 μg/mℓ.

Results

The major findings of this study relate to the levels of peak creative phosphokinase (CPK) present following acute myocardial infarction in infarct patients of the three smoking classes (non-smokers, ex-smokers and recent smokers). This enzyme is released from damaged tissue, and this release correlates well with the extent of myocardial damage. For this analysis three patients were eliminated who had peak CPK values greater than 4,000 IU (International Units) associated with multiple and/or prolonged cardiac resuscitation during the first 24 hours after admission to the hospital.

Figure 1 shows the distribution of peak CPK measurements for the three smoking groups. The number in each CPK class is presented as a percent of the total number in each of three smoking classes (non-smokers, ex-smokers, recent smokers). A relatively large percent of the non-smokers had peak CPK's between 0 and 400 IU and relatively few non-smokers had CPK levels of greater than 1,000 IU. In contrast a relatively small percent of the recent smokers had CPK levels less than 400 IU whereas 20% of the recent smokers had CPK levels greater than 1,600 IU. The ex-smokers appeared to fall between the recent smokers and non-smokers. In Figure 2 the average peak CPK levels for the

three smoking classes are presented. The mean peak CPK was 912±85 IU in recent smokers and 472±100 IU in non-smokers. ($p<.005$) and 655±80 IU in ex-smokers ($p=.05$ versus recent smokers). The incidence of peak CPK values of greater than 1,200 IU (Figure 3) was greater in recent smokers (35.4%) than either non-smokers (6.2%, $p<.01$) or ex-smokers (14%, $p<.05$). These data clearly differentiate recent smokers and non-smokers. In both of these figures the ex-smoker group falls between the recent smoker and non-smoker group but the differences do not reach the level of statistical significance. Ex-smokers were compared with non-smokers and recent smokers in a more detailed fashion utilizing a test for a linear trend in proportions (Snedecor, George W., and William G. Cochran. Statistical Methods. Ames, Iowa: The Iowa State University Press, 1967, pp. 246-247). This type of analysis was undertaken to attempt to use all of the data available to decide whether ex-smokers did in fact differ from both non-smokers and recent smokers. Such analysis for the ex-smokers versus the non-smokers is shown in Figure 4. For this analysis the percent of ex-smokers among the ex- and non-smokers in each of nine peak CPK classes (<200, 200-400, 400-600, 600-800, 800-1,000, 1,000-1,200, 1,200-1,400, 1,400-1,600 and >1,600) was calculated. The regression equation of ex-smokers as a percent of total ex-smokers and non-smokers in these nine peak CPK classes is $y=4.95X + 42.0$, where X=CPK class (one to nine), y=% ex-smokers. The slope of this line is significantly different from zero, ($p < .05$). A similar analysis of recent smokers as a percent of the total recent and ex-smokers group (Figure 5) was performed. The regression equation is $y=3.40X + 44.3$, y=% recent smokers. Again the slope of this relationship is significantly greater than zero, $p < .05$). Thus ex-smokers appear to have a greater incidence of high peak CPK values than non-smokers and a lower incidence than recent smokers; that is, ex-smokers appear to differ from both recent and non-smokers.

We have entertained a number of alternative explanations for these data beside the explanation that recent smoking is a marker for those who are likely to have relatively high peak CPK levels or large acute myocardial infarction. The first concern was that the intensity of recent smoking may be an index of more significant long-term smoking. From the historical smoking data the average mean pack-years of the ex-smokers and recent smokers was determined. Ex-smokers averaged 55.5 ± 5.5 pack-years and the recent smokers 49.2 ± 4.2 pack-years. Thus those with thiocyanate levels greater than 4.0 μg/mℓ (which identify the recent smoker group) did not appear to have a greater long-term cigarette smoking. Since severity of acute myocardial infarction has at times been related to age, sex, and race, the non-smoker and recent smoker groups were analyzed with regard to these factors. The mean age of the patients with acute myocardial infarction in the three groups are shown in Figure 6. The average age of non-smokers was 67.2 ± 11.7 (S.D.) years, ex-smokers 60.8 ± 10.2 years and recent smokers 54.2 ± 10.4 years (Non-smokers versus Ex-smokers, $p<.02$; Non-smokers versus Recent smokers, $p<.001$; and Recent smokers versus Ex-

smokers, $p < .005$). Since recent smokers tended to be younger, the mean peak CPK values were analyzed by age classes in the non-smoker and recent smoker group.

Although the mean peak CPK value for those less than 50 years was slightly higher than those in the 50 to 60 year or greater than 60 year age groups, these differences were not statistically significant (Figure 7). Likewise, in the non-smoker group there was no statistically significant difference between the peak CPK values of those less than or greater than 60 years of age (Figure 8). The nine non-smokers less than 60 years of age had peak value of 310 ± 140 (SE) IU and the 45 recent smokers with an age of less than 60 years had a peak CPK value of 948 + 109 (SE) ($p < .02$). Thus although there were differences in the smoking classes with regard to age, age did not appear to be the determining factor in accounting for the higher mean value among recent smokers. Of the non-smoking group 53.1% were white of the ex-smokers 62.8% were white. Of the recent smokers, 60.0% were white. Thus race did not appear to account for the differences in peak CPK values.

The percent of the male patients in each smoking class is presented in Figure 9. Only 40.6% of the non-smokers were male whereas 70.8% of recent smokers and 86.0% of ex-smokers were male. In Figure 10 the non-smoker and recent smoker groups are divided into male and female and the value of the mean peak CPK in each subgroup presented. There is no difference between the average peak CPK values of male and female subjects in either of the two smoking classes. Thus the differences in age, sex, and race among various smoking classes do not appear to account for the differences in the peak CPK values observed.

To examine the possibility that CPK metabolism may be influenced by the extent of recent cigarette smoking, k_d (fractional disappearance rate of CPK) in eight patients was compared to the thiocyanate level. There was no apparent correlation between the k_d and the intensity of recent smoking as assessed by the thiocyanate level.

Recent smokers with acute myocardial infarction in the EW group had blood carboxyhemoglobin levels of 3.5 ± .4% (n=34). This level was significantly greater than the non-smokers (0.5 ± 0.1% n=16) and than ex-smokers (0.4 ± 1% n=13) with acute infarction. This difference was, of course, expected because of the overwhelming effect of smoking on COHb levels, and the correlation of thiocyanate with COHb. The carboxyhemoglobin level of smokers without acute infarction was 3.0 ± .47%, n=26. The average thiocyanate level of the no infarct group was compared with the total infarct population. There was no significant difference in the average thiocyanate level (infarct group thiocyanate level =4.29 ± .22 µg/mℓ, and the no infarct group thiocyanate level =4.72 ± .41 µg/mℓ; n.s.). Thus the relative intensity of

recent smoking among the infarct population as a whole was not greater than the group of patients seen in the same hospital emergency room with chest pain of uncertain etiology which was not deemed to be due to acute myocardial infarction. The average age of the no infarct group was 59. years (55.8% were male and 61. % were white).

The hemodynamics of patients during the first 24 hours following acute myocardial infarction were studied in the Myocardial Infarction Research Unit in 98 patients whose thiocyanate levels were known. As shown in Table 1, clinical class, stroke work index, radial artery pressure, and cardiac index and heart rate were not related to the smoking class. Left ventricular filling pressure was higher in smokers than ex-smokers (18.0 ± 1.3mm Hg versus 13.9 + 1.2mm Hg, $p < .05$). The ratio of stroke work index (SWI) to left ventricular filling pressure (LVFP) was determined in all patients. A higher ratio would imply that the patient was on a ventricular function curve which is upwards and to the left of patients with a low ratio. (Class IV patients for example would have a low stroke work index and high filling pressure and thus a very low SWI/LVFP). There was no significant difference between smoking classes with regard to SWI/LVFP ratio (Table 1). In Table 2 the incidence of SWI/LVFP of greater than and less than 2.0 g-M/M^2 mmHg in non-smokers, ex-smokers and smokers with thiocyanate levels greater and less than 6 µg/mℓ are shown. In the total of the non-smokers and ex-smokers group, 36% (20/55) of the patients had SWI/LVFP of less than 2.0 implying poor hemodynamic function. In contrast only one of 17 patients with a thiocyanate level of greater than 6 µg/mℓ had a SWI/LVFP of less than 2.0 ($X^2=4.5$, $p<.05$). In Figure 11 the patients with SWI/LVFP of greater than 2.0 are divided into those with thiocyanate levels greater than or less than <6 mcg/mℓ regardless of smoking class and evaluated with regard to the percent of patients with peak CPK greater than 1,000 IU. As shown here the patients with the higher thiocyanate levels had a statistically significantly greater incidence of peak CPK values >1,000 IU than those with thiocyanate levels of 6 µg/mℓ. Thus the group of recent heavy smokers tended to have a relatively high SWI/LVFP (implying relatively better left ventricular function) despite a higher incidence of large infarctions. These data are presented in another fashion in Figure 12. Here stroke work index and left ventricular filling pressure for all patients in the hemodynamic study and plotted. The circled points indicate those patients with the thiocyanate level greater than 6 µg/mℓ. Adjacent to the points are the actual peak CPK values for the patients with greater than 6 µg/mℓ thiocyanate. The heavy recent smokers with relatively high peak CPK's sit predominately in the upper left hand corner of this diagram. There is an absence of these patients in the lower right hand corner of the diagram. One possible explanation for this relatively good left ventricular function despite higher values for the peak CPK is a higher level of sympathetic tone in the heavy smokers. Other investigators have measured the catecholamine level

in the blood of smokers and non-smokers with acute myocardial infarction[3] and found a higher level among smokers.

These data taken as a whole suggest that recent smoking is associated with large infarcts, among the population of patients with acute myocardial infarction. This conclusion does conflict with another study of this question. This earlier study did not include objective measures of either recent smoking intensity or objective indices of infarct size[4]. The predominence of large infarcts among the recent smokers did not appear to be attributable to age, sex, or race of the recent smokers nor to the long-term smoking intensity of the recent smokers. The hemodynamic studies during the first 24 hours after admission suggest that heaviest recent smokers had no worse left ventricular function despite higher CPK values possible as a result of increased sympathetic tone.

Survival rates during hospitalization were determined by smoking class. Patients with probable acute infarction but dying prior to obtaining data necessary for strict classification and those with high CPK's associated with cardiac arrest were included in the survival data. Ex-smokers and non-smokers were grouped together since smoking histories were not obtained on the majority of the patients who died shortly after admission with low thiocyanate levels. Of 72 recent smokers 16.7% died where as 18.9% of 90 non-smokers and ex-smokers died. This similar mortality between the groups is present despite the significantly younger age of the smoker group (55.3 ± 11.0 versus 63 ± 11.2, $p < .001$). As in other groups mortality increased with increasing age. In the entire group of 162 patients, 74 patients <60 years of age had a mortality of 4.1%, whereas 86 patients >60 years had a mortality of 31.4%. Among the patients over 60 years of age 29.0% (18/62) non-smokers or former smokers died whereas 40.0% (10/25) recent smokers died. These results suggest that a higher fatality rate may be associated with the larger infarct size.

These studies do not prove that recent smoking causes large infarcts nor that discontinuing smoking may reduce the likelihood of a large infarct. They only serve to identify recent smokers as being a group at high risk for large acute myocardial infarcts among the infarct population. If recent smoking is in fact a causative factor of larger infarcts among smokers (rather than personality type, etc.) the greater size of the infarct may be due to such smoking-related factors as increases in carbon monoxide level in the blood, nicotine, the higher catecholamine levels in such patients or combinations of these and possibly other factors. The fact that ex-smokers had results intermediate between non-smokers and current smokers is consistent with a long-term or chronic effect of smoking independent of the acute effect of increased COHb, nicotine or other smoke products.

Summary

1. Evaluation of the clinical course, indices of the severity of infarction, and hemodynamic changes in patients admitted with chest pain at the Johns Hopkins Hospital has been made in relation to smoking intensity as determined by measurements of carboxyhemoglobin and thiocyanate on admission. Smoking has been evaluated as an indication of CO exposure because it is the principc source of such exposure in Baltimore.

2. Among 140 confirmed cases of myocardial infarction recent or current smokers had evidence of larger infarcts than non-smokers with ex-smokers intermediate. This difference could not be explained by differences in sex, age and race in the three groups.

3. When age is taken into account, the recent smokers had higher mortality than the non-smokers or ex-smokers.

4. Hemodynamic function was relatively good in recent smokers, despite their larger infarcts, perhaps due to catecholamine release associated with smoking.

5. These results are consistent with both an acute and chronic effect on the myocardium of long-term low-level exposures to CO, but clearly cannot be related to that factor by itself. Other constituents of cigarette smoke may be of equal or greater importance in these effects than CO.

6. Because of the relative importance of smoking as a source of CO exposure in the general population, it is unlikely that research on the effects of low-level CO exposure in man can be carried further by this type of study. A more promising group to study would be large groups occupationally exposed to CO alone, in whom the role of cigarette smoking could be assessed independently of CO exposure.

TABLE 1

HEMODYNAMIC MEASUREMENTS IN RECENT, EX, AND NON-SMOKERS WITH ACUTE MYOCARDIAL INFARCTION

	HR (beats/min)	Cardiac Index ($L/min/M^2$)	Radial Art. Press (mmHg) Systolic	Diastolic	LVFP† (mmHg)	SWI** ($g\text{-}M/M^2/mmHg$)	SWI/ LVFP	Severity of Infarction Class II-IV
Recent Smokers (41)	84.5 ± 2.8	2.5 ± .1	148 ± 5	84 ± 3	18.0 ± 1.3*	47.7 ± 3.8	3.92 ± .60	70.7
Ex-smokers (38)	80.11 ± 2.6	2.3 ± .1	154 ± 5	79 ± 3	13.9 ± 1.2	47.1 ± 3.6	5.13 ± .87	47.5
Non-smokers (18)	82.2 ± 5.8	2.2 ± .2	138 ± 9	72 ± 4	15.1 ± 2.0	43.1 ± 6.5	5.47 ± 1.8	55.6

*$p < .05$ versus ex-smokers.
†Left ventricular Filling Pressure
**Stroke Work Index

TABLE II

SWI/LVFP IN MIRU PATIENTS

	Non Smokers	Ex-Smokers	SCN < 6 µg/mℓ	SCN > 6 µg/mℓ*
SWI/LVFP < 2.0 (g-M/M^2)	8	12	10	1
SWI/LVFP > 2.0 (g-M/M^2)	9	26	10	16

*$p < .05$ versus total of non and ex-smokers

FIGURE 1

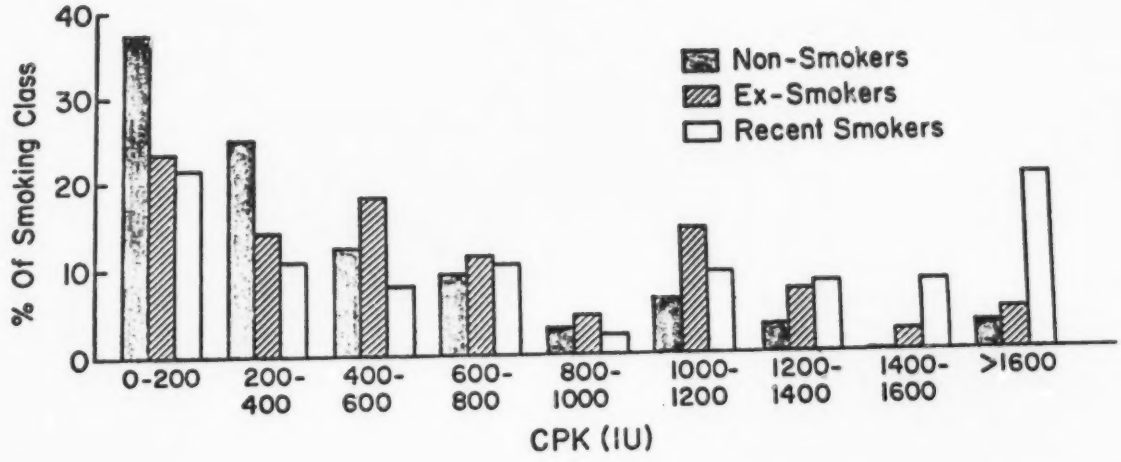

Figure 1: The percent of 65 recent smokers, 43 smokers and 32 non-smokers with acute myocardial infarction in each of nine peak CPK classes.

FIGURE 2

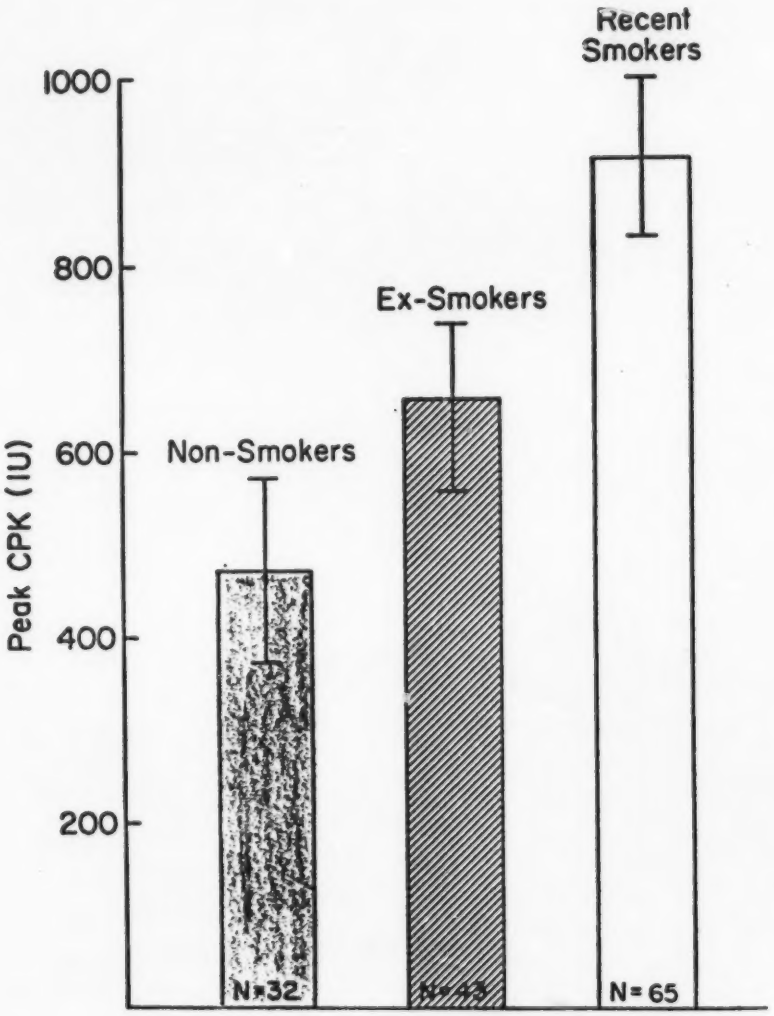

Figure 2: Peak CPK in three smoking classes. Bars = S.E.M. Recent versus Non-smokers, $p < .005$.

FIGURE 3

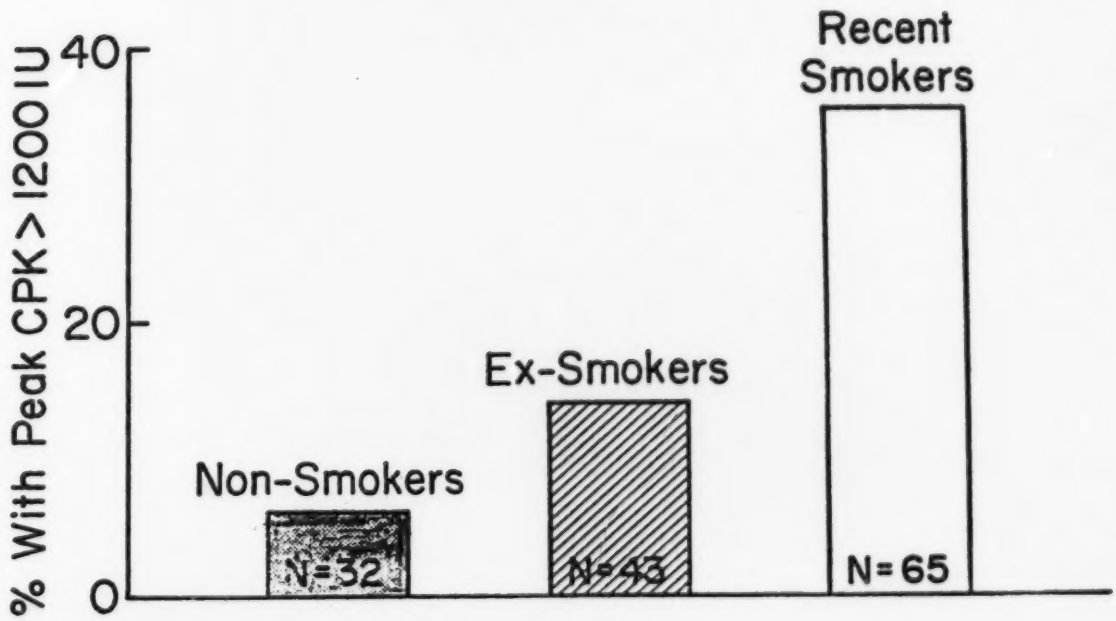

Figure 3: Incidence of peak CPK > 1,200 IU in three smoking classes. Recent smokers versus non smokers $p < .01$; Recent smokers versus Ex-smokers, $p < .05$.

FIGURE 4

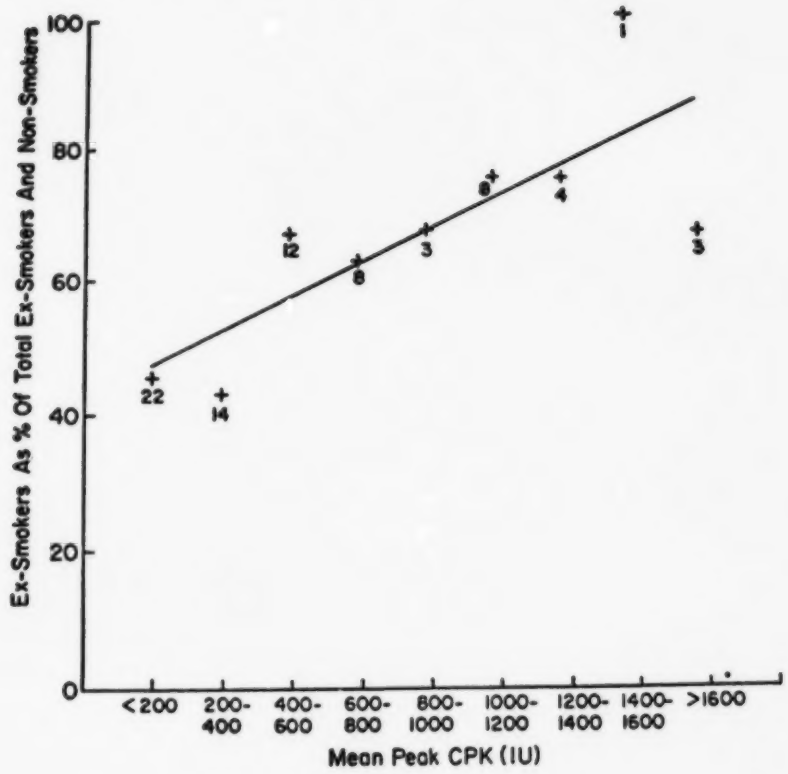

Figure 4: Ex-smokers and non-smokers with infarcts examined in terms of peak CPK using a linear trend in proportions analysis (see text). Small numbers indicate total number of ex and non smokers in each CPK class.

FIGURE 5

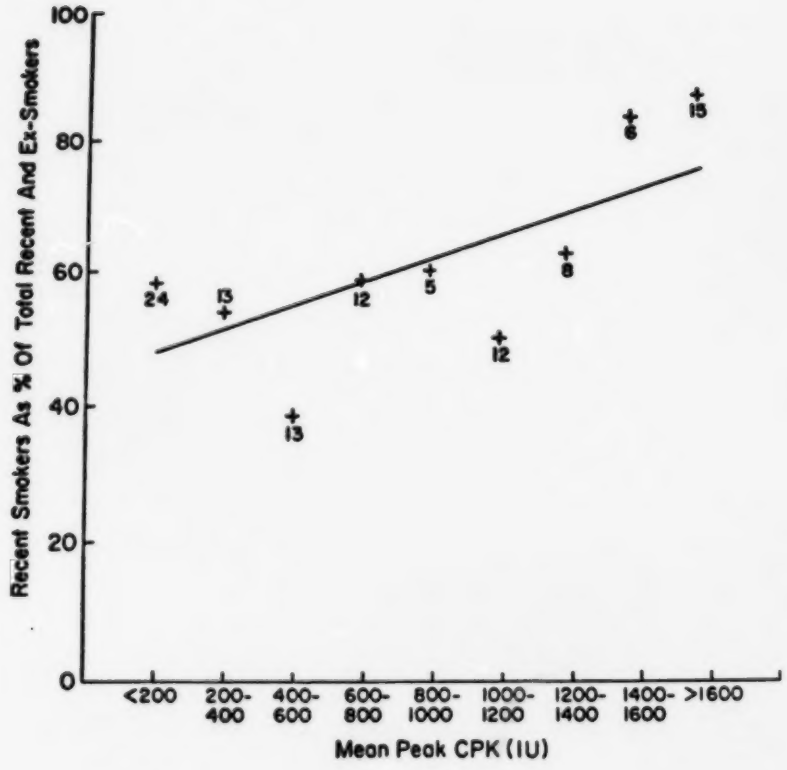

Figure 5: Recent smokers and ex-smokers with infarcts examined in terms of peak CPK using a linear trend in proportions analysis (see text). Small numbers indicate the total number of recent and ex-smokers in each CPK class.

FIGURE 6

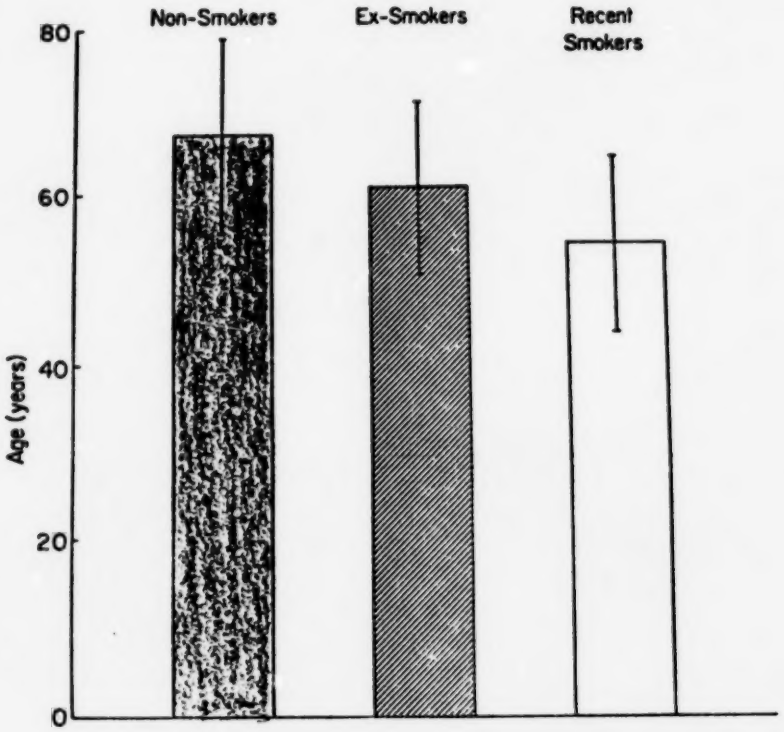

Figure 6: Age of infarct patients in each smoking class. Bars = S.D.

FIGURE 7

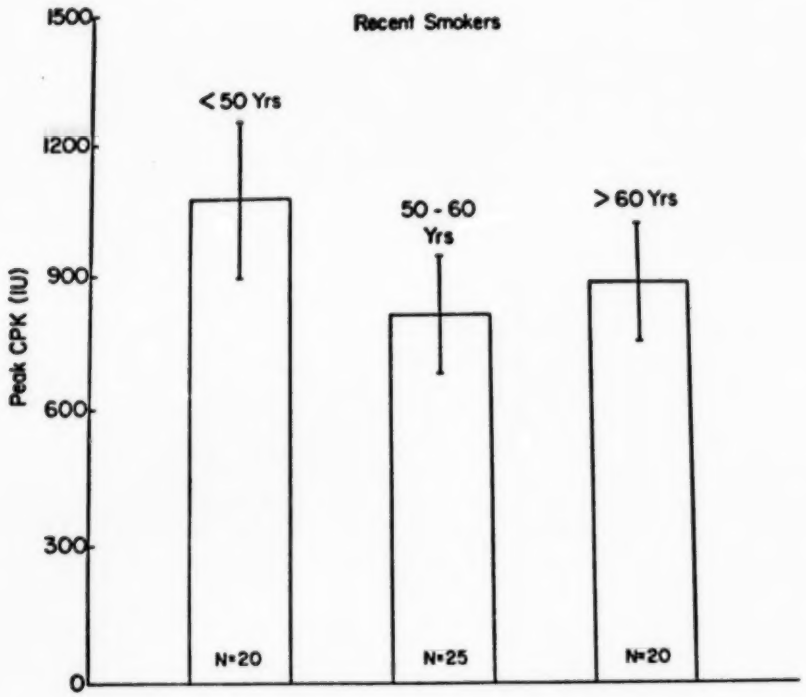

Figure 7: Peak CPK by age class in recent smokers with acute myocardial infarctions. Bars = S.E.M.

FIGURE 8

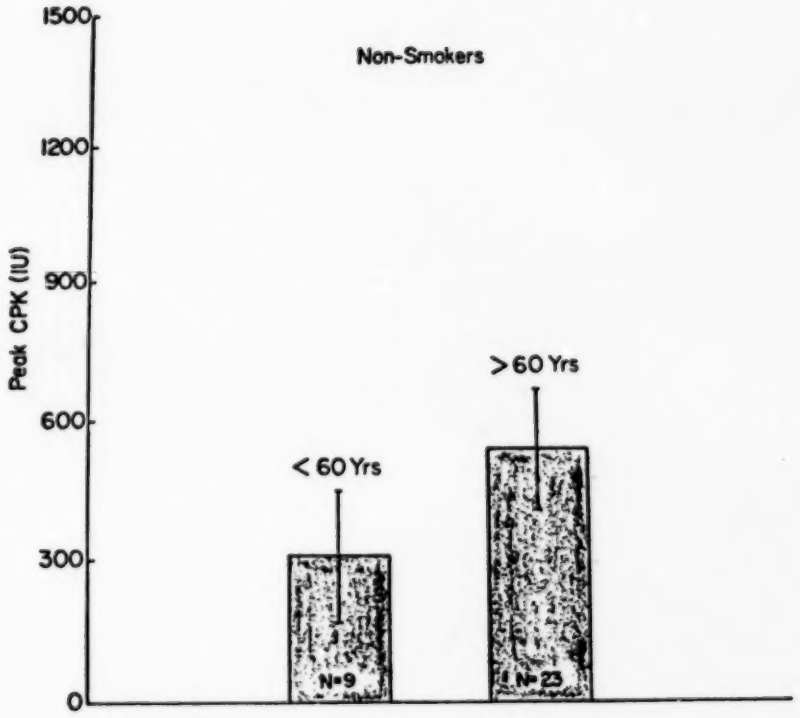

Figure 8: Peak CPK by age class in non-smokers with acute myocardial infarction. Bars = S.E.M.

FIGURE 9

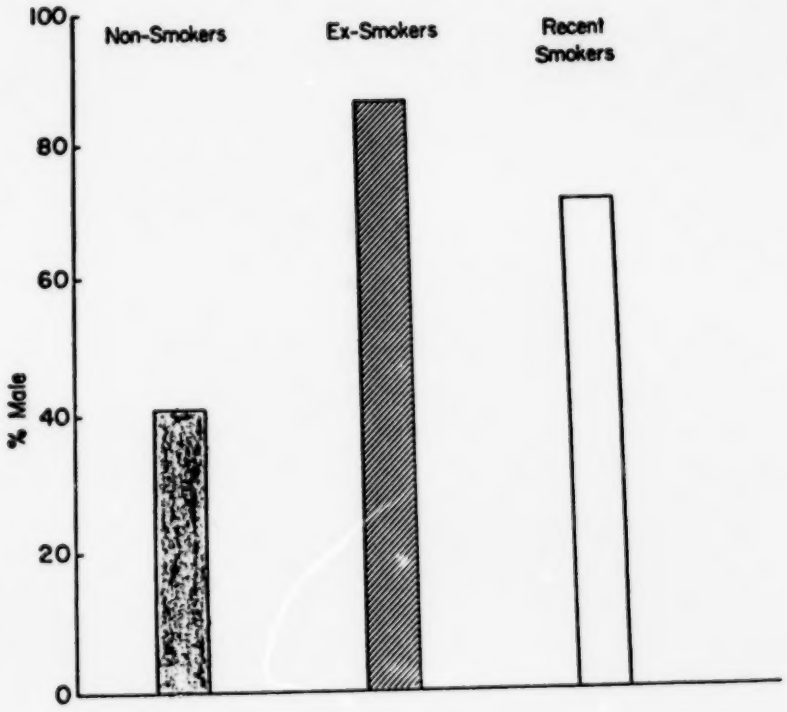

Figure 9: Percent male patients with acute myocardial infarction in each smoking class.

FIGURE 10

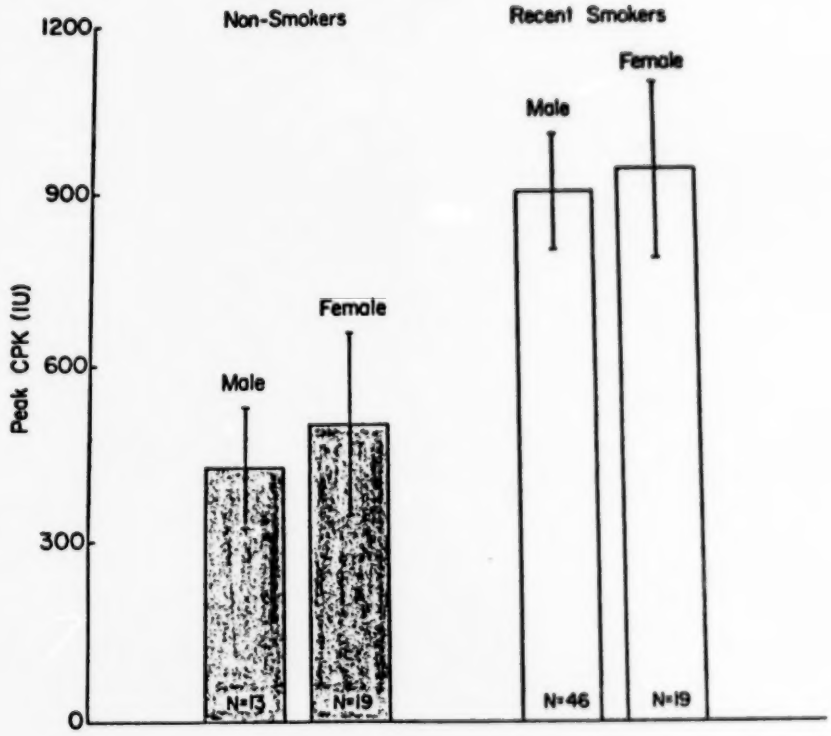

Figure 10: Peak CPK by sex among recent smokers and non-smokers with acute myocardial infarction. Bars = S.E.M.

FIGURE 11

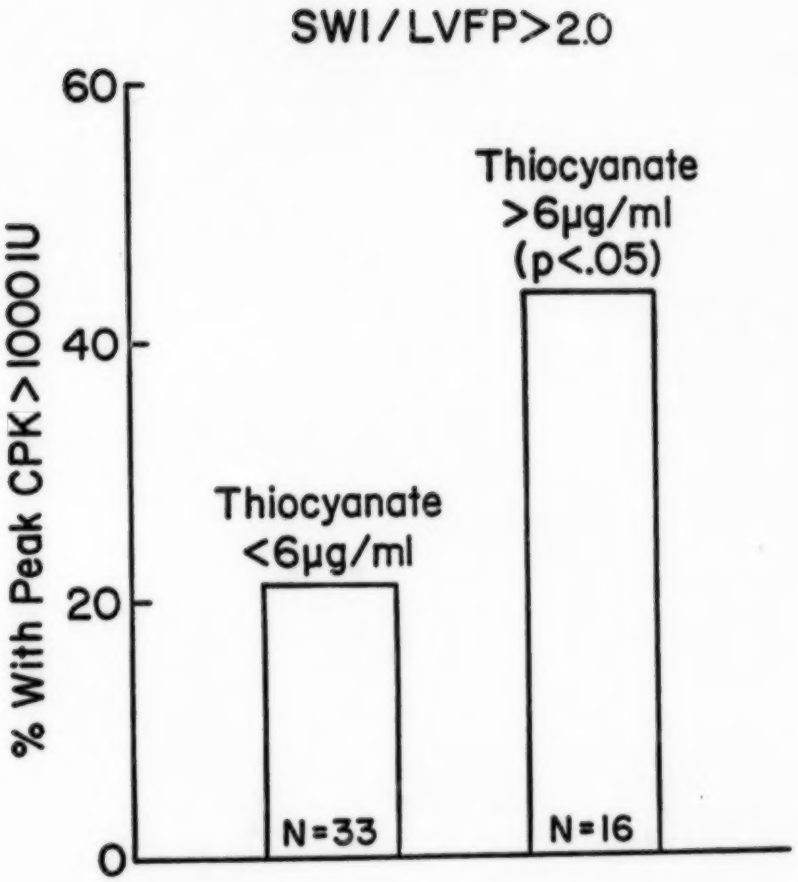

Figure 11: Incidence of peak CPK > 1,000 IU in patients with acute myocardial infarction whose SWI/LVFP > 2.0 g-M/M^2 mmHg divided in to those with thiocyanates > 6 μg/mℓ and thiocyanates < 6 μg/mℓ.

FIGURE 12

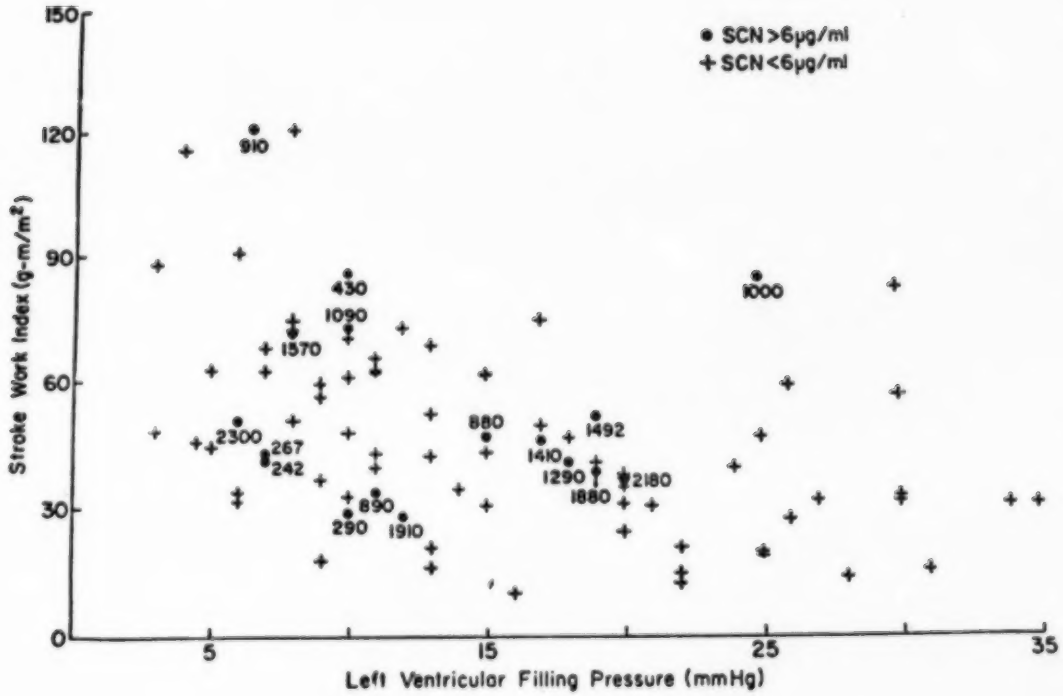

Figure 12: All infarct MIRU patients plotted by SWI and LVFP: Numbers indicate peak CPK of patients with thiocyanate levels (SCN > 6 μg/ml.

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